



## U.S. Environmental Protection Agency Integrated Risk Information System

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### Aldrin (CASRN 309-00-2)

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#### Aldrin; CASRN 309-00-2

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

#### STATUS OF DATA FOR Aldrin

File First On-Line 03/31/1987

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	on-line	03/01/1988
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	on-line	07/01/1993

#### I. Chronic Health Hazard Assessments for Noncarcinogenic Effects

##### I.A. Reference Dose for Chronic Oral Exposure (RfD)

Substance Name -- Aldrin  
CASRN -- 309-00-2  
Last Revised -- 03/01/1988

The oral Reference Dose (RfD) is based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. It is expressed in units of mg/kg-day. In general, the RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Please refer to the Background Document for an elaboration of these concepts. RfDs can also be derived for the noncarcinogenic health effects of substances that are also carcinogens. Therefore, it is essential to refer to other

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sources of information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

[Quantitative Estimate of Carcinogenic Risk from Oral Exposure](#)

#### I.A.1. Oral RfD Summary

Critical Effect	Experimental Doses*	UF	MF	RfD
Liver toxicity	NOAEL: none	1000	1	3E-5 mg/kg/day
Rat Chronic Feeding Study	LOAEL: 0.5 ppm diet (0.025 mg/kg/day)			
Fitzhugh et al., 1964				

- [Summary of Risk Estimates](#)  
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\* Conversion Factors: 1 ppm = 0.05 mg/kg/day (assumed rat food consumption)

#### I.A.2. Principal and Supporting Studies (Oral RfD)

Fitzhugh, O.G., A.A. Nelson, and M.L. Quaife. 1964. Chronic oral toxicity of aldrin and dieldrin in rats and dogs. Food Cosmet. Toxicol. 2: 551-562.

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Groups of 24 rats (12/sex) were fed aldrin in the diet at levels of 0, 0.5, 2, 10, 50, 100, or 150 ppm for 2 years. Liver lesions characteristic of chlorinated insecticide poisoning were observed at dose levels of 0.5 ppm and greater. These lesions were characterized by enlarged centrilobular hepatic cells, with increased cytoplasmic oxyphilia, and peripheral migration of basophilic granules. A statistically significant increase in liver-to-body weight ratio was observed at all dose levels. Kidney lesions occurred at the highest dose levels. Survival was markedly decreased at dose levels of 50 ppm and greater.

Additional data are fairly supportive. Effect and no-effect levels are similar (to those found for rats) for liver effects in dogs after 15 months' exposure to aldrin in the diet. Liver effects were observed at slightly higher doses in several other subchronic-to-chronic rat and dog studies. Short-term exposure to higher doses resulted in mortality for a number of species.

#### I.A.3. Uncertainty and Modifying Factors (Oral RfD)

UF -- The composite UF of 1000 encompasses the uncertainty of extrapolation from animals to humans, the uncertainty in the range of human sensitivities, and an additional uncertainty because the RfD is based on a LOAEL rather than a NOAEL.

MF -- None

#### I.A.4. Additional Studies/Comments (Oral RfD)

None.

#### I.A.5. Confidence in the Oral RfD

Study -- Medium  
Database -- Medium  
RfD -- Medium

The principal study, designed as a carcinogenesis bioassay, is strong in histopathologic analysis but lacks other toxicologic parameters, and is therefore rated medium. The database is fairly extensive, and generally supportive, but is rated medium because of the lack of NOELs for some studies. Also, no chronic data exist for the dog, which may be a more sensitive species than the rat. Medium confidence in the RfD follows.

#### **\_\_I.A.6. EPA Documentation and Review of the Oral RfD**

U.S. EPA. 1982. Toxicity-Based Protective Ambient Water Levels for Various Carcinogens. Environmental Criteria and Assessment Office, Cincinnati, OH. ECAO-CIN-431. Internal review draft.

The RfD has been reviewed internally by ECAO-Cin.

Agency Work Group Review -- 12/18/1985

Verification Date -- 12/18/1985

Screening-Level Literature Review Findings -- A screening-level review conducted by an EPA contractor of the more recent toxicology literature pertinent to the RfD for Aldrin conducted in September 2002 did not identify any critical new studies. IRIS users who know of important new studies may provide that information to the IRIS Hotline at [hotline.iris@epa.gov](mailto:hotline.iris@epa.gov) or (202)566-1676.

#### **\_\_I.A.7. EPA Contacts (Oral RfD)**

Please contact the IRIS Hotline for all questions concerning this assessment or IRIS, in general, at (202)566-1676 (phone), (202)566-1749 (FAX) or [hotline.iris@epa.gov](mailto:hotline.iris@epa.gov) (internet address).

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#### **\_\_I.B. Reference Concentration for Chronic Inhalation Exposure (RfC)**

Substance Name -- Aldrin  
CASRN -- 309-00-2

Not available at this time.

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### **\_\_II. Carcinogenicity Assessment for Lifetime Exposure**

Substance Name -- Aldrin  
CASRN -- 309-00-2  
Last Revised -- 07/01/1993

Section II provides information on three aspects of the carcinogenic assessment for the substance in question; the weight-of-evidence judgment of the likelihood that the substance is a human carcinogen, and quantitative estimates of risk from oral exposure and from inhalation exposure. The quantitative risk estimates are

presented in three ways. The slope factor is the result of application of a low-dose extrapolation procedure and is presented as the risk per (mg/kg)/day. The unit risk is the quantitative estimate in terms of either risk per ug/L drinking water or risk per ug/cu.m air breathed. The third form in which risk is presented is a drinking water or air concentration providing cancer risks of 1 in 10,000, 1 in 100,000 or 1 in 1,000,000. The rationale and methods used to develop the carcinogenicity information in IRIS are described in The Risk Assessment Guidelines of 1986 (EPA/600/8-87/045) and in the IRIS Background Document. IRIS summaries developed since the publication of EPA's more recent Proposed Guidelines for Carcinogen Risk Assessment also utilize those Guidelines where indicated (Federal Register 61(79):17960-18011, April 23, 1996). Users are referred to Section I of this IRIS file for information on long-term toxic effects other than carcinogenicity.

## **\_\_II.A. Evidence for Human Carcinogenicity**

### **\_\_II.A.1. Weight-of-Evidence Characterization**

Classification -- B2; probable human carcinogen

Basis -- Orally administered aldrin produced significant increases in tumor responses in three different strains of mice in both males and females. Tumor induction has been observed for structurally related chemicals, including dieldrin, a metabolite.

### **\_\_II.A.2. Human Carcinogenicity Data**

Inadequate. Two studies of workers exposed to aldrin and dieldrin (a metabolite of aldrin) did not find these workers to have an excess risk of cancer. Both studies, however, were limited in their ability to detect an excess of deaths from cancer. Van Raalte (1977) observed two cases of cancer (gastric and lymphosarcoma) among 166 pesticide manufacturing workers exposed 4 to 19 years and followed from 15 to 20 years. Exposure was not quantified, and workers were also exposed to other organochlorine pesticides (endrin and telodrin). A small number of workers was studied, the mean age of the cohort (47.7 years) was low, the number of expected deaths was not calculated, and the duration of exposure and of latency was relatively short.

In a retrospective mortality study, Ditraglia et al. (1981) reported no increased incidence of deaths from cancer among 1155 organochlorine pesticide manufacturing workers (31 observed vs. 37.8 expected, SMR=82). This result was not statistically significant. Workers were employed for 6 or more months and followed for 13 or more years (24,939 person-years). Workers with no exposure (for example, office workers) were included in the cohort. Vital status was not known for 112 (10%) of the workers, and these workers were assumed to be alive; therefore, additional deaths may have occurred but were not observed. Exposure was not quantified and workers were also exposed to other chemicals and pesticides (including endrin). An increased incidence of deaths from cancer was seen at several specific sites: esophagus (2 deaths observed, SMR=235), rectum (3, SMR=242); liver (2, SMR=225), and lymphatic and hematopoietic system (6, SMR=147); but these site-specific incidences were not statistically significant.

### **\_\_II.A.3. Animal Carcinogenicity Data**

Sufficient. Davis and Fitzhugh (1962) fed a group of 215 male and female C3HeB/Fe mice a dietary mixture containing 10 ppm aldrin for up to 2 years. The control group consisted of 217 mice. The aldrin-treated mice died 2 months earlier than controls. Intercurrent disease, pneumonia, and intestinal parasitism may have influenced the long-term survival rate. A statistically significant increase of hepatomas was reported in the treated animals as compared with controls. An independent reevaluation of the

liver lesions showed most of the hepatomas to be liver carcinomas (Epstein, 1975). In a follow-up study, Davis (1965) administered aldrin at 0 or 10 ppm in the diet to 100 male and 100 female C3H mice for 2 years. The incidence of hepatic hyperplasia and benign hepatomas in the aldrin group was approximately double that of controls, whereas the number of hepatic carcinomas was about the same. Neither study provided a detailed pathologic examination or data separated by sex.

Aldrin (95% pure) was administered in the diet to 50 male and 50 female B6C3F1 mice at TWA doses of 4 and 8 ppm or 3 and 6 ppm. Treatment was for 80 weeks, and animals were observed for an additional 10 to 13 weeks (NCI, 1978). In male mice, there was a significant dose-related increase in hepatocellular carcinomas when compared with matched or pooled controls.

Treon and Cleveland (1955) administered aldrin in the diet to 40 Carworth rats/sex at concentrations of 2.5, 12.5, or 25 ppm for a period of 2 years. Forty animals/sex served as controls. Mortality of the treated rats was greater than controls, with 50% surviving in the 2.5 and 12.5 ppm groups and 40% surviving in the 25 ppm group at the end of the experiment. Cleveland (1966) reported that no apparent treatment-related tumors were present in the above study. Deichmann et al. (1970) fed 50 male and 50 female Osborne-Mendel rats aldrin (95% pure) at final concentrations of 20, 30, or 50 ppm for 31 months. Controls consisted of 100 rats/sex. There was no evidence of carcinogenic response in male or female rats fed aldrin. The NCI (1978) fed 50 Osborne-Mendel rats/sex aldrin (95% pure) at 30 or 60 ppm. Male rats were treated 111 to 113 weeks and followed for 37 to 38 weeks of observation, and female rats were treated for 80 weeks and followed for 32 to 33 weeks of observation. Aldrin produced no significant effect on the mortality of the rats of either sex. The tumors observed were randomly distributed, with no apparent relationship to aldrin treatment. Four additional bioassays observed no carcinogenic effect of aldrin in rats, but were considered inadequate for carcinogenicity assessment.

#### II.A.4. Supporting Data for Carcinogenicity

Aldrin causes chromosomal aberrations in mouse, rat, and human cells (Georgian, 1974) and unscheduled DNA synthesis in rats (Probst et al., 1981) and humans (Rocchi et al., 1980) cells. Aldrin does not cause reverse mutations in *S. typhimurium*, *E. coli*, or *S. marcescens*, or mitotic gene conversion in *S. cerevisiae* (Fahrig, 1974).

Five compounds structurally related to aldrin--dieldrin, chlordane, heptachlor, heptachlor epoxide, and chlorendic acid--have induced malignant liver tumors in mice. Chlorendic acid has also induced liver tumors in rats.

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#### II.B. Quantitative Estimate of Carcinogenic Risk from Oral Exposure

##### II.B.1. Summary of Risk Estimates

Oral Slope Factor --  $1.7 \times 10^1$  per (mg/kg)/day

Drinking Water Unit Risk --  $4.9 \times 10^{-4}$  per (ug/L)

Extrapolation Method -- Linearized multistage procedure, extra risk

## Drinking Water Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	2E-1 ug/L
E-5 (1 in 100,000)	2E-2 ug/L
E-6 (1 in 1,000,000)	2E-3 ug/L

**\_\_II.B.2. Dose-Response Data (Carcinogenicity, Oral Exposure)**

Tumor Type -- liver carcinoma

Test animals -- mouse/C3H (Davis); mouse/B6C3F1, male (NCI)

Route -- diet

Reference -- Davis, 1965 (see table); NCI, 1978

Administered Dose (ppm)	Human Equivalent Dose (mg/kg-day)	Tumor Incidence	Reference
females			
0	0	2/53	Davis, 1965 reevaluated by Reuber (cited in Epstein, 1975)
10	0.104	72/85	
males			
0	0	22/73	
10	0.104	75/91	
0	0	3/20	NCI, 1978
4	0.04	16/49	
8	0.08	25/45	

**\_\_II.B.3. Additional Comments (Carcinogenicity, Oral Exposure)**

Body weights for mice were assumed to be 0.03 kg for purposes of dose conversion. The above data sets were used for calculation of the following slope factors: 2.3E+1 per (mg/kg)/day for female C3H mice, 1.8E+1 per (mg/kg)/day for male C3H mice, and 1.2E+1 per (mg/kg)/day for male B6C3F1 mice. No strain or sex specificity was noted in the studies, since aldrin treatment induced liver tumors in all mouse strains tested. A geometric mean of 1.7E+1 per (mg/kg)/day was thus chosen for the quantitative estimate, since all three slope factors were very similar.

The unit risk should not be used if the water concentration exceeds 20 ug/L, since above this concentration the unit risk may not be appropriate.

**\_\_II.B.4. Discussion of Confidence (Carcinogenicity, Oral Exposure)**

Adequate numbers of animals were treated for a large proportion of their lifetime. The route of treatment was appropriate. Slope factors calculated from three data sets from two independent assays were within a factor of 2. A slope factor for dieldrin, a major metabolite of aldrin, was determined to be 1.6E+1, essentially identical to that of aldrin.

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**\_\_II.C. Quantitative Estimate of Carcinogenic Risk from Inhalation Exposure**

**\_\_II.C.1. Summary of Risk Estimates**

Inhalation Unit Risk -- 4.9E-3 per (ug/cu.m)

Extrapolation Method -- Linearized multistage procedure, extra risk

Air Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	2E-2 ug/cu.m
E-5 (1 in 100,000)	2E-3 ug/cu.m
E-6 (1 in 1,000,000)	2E-4 ug/cu.m

**\_\_II.C.2. Dose-Response Data for Carcinogenicity, Inhalation Exposure**

The unit risk was calculated from the oral data presented in II.B.2.

**\_\_II.C.3. Additional Comments (Carcinogenicity, Inhalation Exposure)**

The unit risk should not be used if the air concentration exceeds 2 ug/cu.m, since above this concentration the unit risk may not be appropriate.

**\_\_II.C.4. Discussion of Confidence (Carcinogenicity, Inhalation Exposure)**

See II.B.4.

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**\_\_II.D. EPA Documentation, Review, and Contacts (Carcinogenicity Assessment)****\_\_II.D.1. EPA Documentation**

Source Document -- U.S. EPA, 1986

The values in the 1986 Carcinogenicity Assessment for Aldrin/Dieldrin have been reviewed by the Carcinogen Assessment Group.

**\_\_II.D.2. EPA Review (Carcinogenicity Assessment)**

Agency Work Group Review -- 03/22/1987

Verification Date -- 03/22/1987

Screening-Level Literature Review Findings -- A screening-level review conducted by an EPA contractor of the more recent toxicology literature pertinent to the cancer assessment for Aldrin conducted in September 2002 identified one or more significant new studies. IRIS users may request the references for those studies from the IRIS Hotline at [hotline.iris@epa.gov](mailto:hotline.iris@epa.gov) or (202)566-1676.

**\_\_II.D.3. EPA Contacts (Carcinogenicity Assessment)**



Please contact the IRIS Hotline for all questions concerning this assessment or IRIS, in general, at (202)566-1676 (phone), (202)566-1749 (FAX) or [hotline.iris@epa.gov](mailto:hotline.iris@epa.gov) (internet address).

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\_III. [reserved]

\_IV. [reserved]

\_V. [reserved]

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## \_VI. Bibliography

Substance Name -- Aldrin  
CASRN -- 309-00-2  
Last Revised -- 09/01/1989

### \_VI.A. Oral RfD References

Fitzhugh, O.G., A.A. Nelson, and M.L. Quaife. 1964. Chronic oral toxicity of aldrin and dieldrin in rats and dogs. Food Cosmet. Toxicol. 2: 551-562.

U.S. EPA. 1982. Toxicity-Based Protective Ambient Water Levels for Various Carcinogens. Environmental Criteria and Assessment Office, Cincinnati, OH. ECAO-CIN-431. Internal review draft.

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### \_VI.B. Inhalation RfC References

None

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### \_VI.C. Carcinogenicity Assessment References

Cleveland, F.P. 1966. A summary of work on aldrin and dieldrin toxicity at the Kettering Laboratory. Arch. Environ. Health. 13: 195.

Davis, K.J. 1965. Pathology report on mice fed dieldrin, aldrin, heptachlor, or heptachlor epoxide for two years. Internal FDA memorandum to Dr. A.J. Lehrman, July 19.

Davis, K.J. and O.G. Fitzhugh. 1962. Tumorigenic potential of aldrin and dieldrin for mice. Toxicol. Appl. Pharmacol. 4: 187-189.

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Epstein, S.S. 1975. The carcinogenicity of dieldrin. Part 1. *Sci. Total Environ*. 4: 1-52.

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Georgian, L. 1975. The comparative cytogenic effects of aldrin and phosphamidon. *Mutat. Res*. 31: 103-108.

NCI (National Cancer Institute). 1978. Bioassays of aldrin and dieldrin for possible carcinogenicity. DHEW Publication No. (NIH) 78-821. *NCI Carcinogenesis Tech. Rep. Ser. No. 21. NCI-C6-TR-21*.

Probst, G.S., R.E. McMahon, L.W. Hill, D.Z. Thompson, J.K. Epp and S.B. Neal. 1981. Chemically-induced unscheduled DNA synthesis in primary rat hepatocyte cultures: A comparison with bacterial mutagenicity using 218 chemicals. *Environ. Mutagen*. 3: 11-32.

Rocchi, P., P. Perocco, W. Alberghini, A. Fini and G. Prodi. 1980. Effect of pesticides on scheduled and unscheduled DNA synthesis of rat thymocytes and human lymphocytes. *Arch. Toxicol*. 45: 101-108.

Treon, J.F. and F.P. Cleveland. 1955. Toxicity of certain chlorinated hydrocarbon insecticides for laboratory animals, with special reference to aldrin and dieldrin. *Agric. Food Chem*. 3: 402-408.

U.S. EPA. 1986. Carcinogenicity Assessment of Aldrin and Dieldrin. Prepared by the Office of Health and Environmental Assessment, Carcinogen Assessment Group, Washington, DC, for the Hazard Evaluation Division, Office of Pesticides and Toxic Substances, Office of Pesticide Programs, Washington, DC.

Van Raalte, H.G.S. 1977. Human experience with dieldrin in perspective. *Ecotoxicol. Environ. Safety*. 1: 203-210.

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## **\_VII. Revision History**

Substance Name -- Aldrin  
CASRN -- 309-00-2

Date	Section	Description
09/30/1987	II.	Carcinogenicity section added
03/01/1988	II.B.4.	Confidence statement revised
12/01/1988	II.B.4.	Corrected slope factor in text
09/01/1989	II.A.2.	Ditraglia reference changed to Ditraglia et al.
09/01/1989	II.A.3.	Deichmann reference changed to Deichmann et al.
09/01/1989	II.B.3.	Body weight for mice corrected to kg

09/01/1989	VI.	Bibliography on-line
01/01/1991	II.	Text edited
01/01/1991	II.C.1.	Inhalation slope factor removed (global change)
01/01/1992	I.A.7.	Secondary contact changed
01/01/1992	IV.	Regulatory actions updated
07/01/1993	II.D.3.	Secondary contact's phone number changed
04/01/1997	III., IV., V.	Drinking Water Health Advisories, EPA Regulatory Actions, and Supplementary Data were removed from IRIS on or before April 1997. IRIS users were directed to the appropriate EPA Program Offices for this information.
12/03/2002	I.A.6., II.D.2.	Screening-Level Literature Review Findings message has been added.

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## **\_VIII. Synonyms**

Substance Name -- Aldrin  
CASRN -- 309-00-2  
Last Revised -- 03/31/1987

309-00-2

Aldrex

Aldrin

Aldrite

Aldrosol

1,4:5,8-Dimethanonaphthalene, 1,2,3,4,10,10-Hexachloro-1,4,4a,5,8,8a-Hexahydro-, (1 alpha, 4 alpha, 4a beta, 5 alpha, 8 alpha, 8a beta)-

1,4:5,8-Dimethanonaphthalene, 1,2,3,4,10,10-Hexachloro-1,4,4a,5,8,8a-Hexahydro-

Drinox

ENT 15,949

1,2,3,4,10,10-Hexachloro-1,4,4a,5,8,8a-Hexahydro-1,4,5,8-Dimethanonaphthalene

1,2,3,4,10,10-Hexachloro-1,4,4a,5,8,8a-Hexahydro-1,4-endo-exo-5,8-Dimethanonaphthalene

1,2,3,4,10,10-Hexachloro-1,4,4a,5,8,8a-Hexahydro-exo-1,4-endo-5,8-Dimethanonaphthalene

Hexachlorohexahydro-endo-exo-Dimethanonaphthalene

HHDN

NCI-C00044

Octalene

Seedrin

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